Causality-Bettie, txt

: (chronic disease [MH] OR neoplasms [MH] OR cancer OR asthma[MH] OR pulmonary emphysema [MH] hysema [MH]) AND (tobacco [MH] OR smoking [MH] OR tobacco use disorder [MH]) AND (causality [MH] iration [MH] OR inhalation OR "early detection" OR "menthol" OR "unfiltered cigarettes" OR iltered cigarettes" OR "filter cigarettes" OR "pattern of use" OR "early diagnosis")

Epidemiol 1999 Feb; 9(2):114-20

lated cigarette smoking and lung-cancer risk.

ter CL, Jarvik ME, Morgenstern H, McCarthy WJ, London SJ

ment of Preventive Medicine, USC School of Medicine, Norris Comprehensive Center, Los Angeles, CA 90033, USA.

E: Menthol smoking may lead to a greater increase in lung-cancer risk than g of nonmentholated cigarettes. Mentholation of cigarettes adds additional ogenic components to cigarette smoke and increases retention times for tte smoke in the lungs. Only two epidemiologic studies have been conducted thol smoking and lung cancer, and their results are conflicting. Of note, n American males have much higher rates of lung cancer than Caucasian despite smoking fewer cigarettes per day. Because the consumption of 1 cigarettes is much more frequent among African Americans, it is of st to examine the possible association between menthol smoking and ancer risk in this population. METHODS: We examined the association n menthol cigarette smoking and lung-cancer risk among smokers by ing 337 incident cases of lung cancer with 478 population controls ed in a case-control study of lung cancer. Information on smoking history her known and potential risk factors for lung cancer, including dietary, was obtained by in-person interviews. RESULTS: The adjusted odds ratios t differ appreciably between smokers of mentholated cigarettes versus ive nonmentholated cigarette smokers in the overall study group of s. The odds ratio (OR) for 32 pack-years or more of mentholated vs. tholated cigarettes was 0.90 (95% confidence interval (CI) = 0.38-2.12) in n Americans and 1.06 (95% CI = 0.47-2.36) in Caucasians, and did not for either ethnic group (p = 0.98). CONCLUSIONS: Our results suggest that ng-cancer risk from smoking mentholated cigarettes resembles the risk from g non-mentholated cigarettes. Our data do not support the hypothesis that creased risk of lung cancer among African Americans is due to the sed prevalence of menthol smoking.

10037555, UI; 99155159

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9337294, UI: 97477231

ev Med 1997 Jul-Aug; 26(4): 451-6

t of filter cigarette smoking on lung cancer histology.

man SD, Muscat JE, Hoffmann D, Wynder EL

ion of Epidemiology, American Health Foundation, New York, New York 10017,

ROUND: The rates of lung adenocarcinoma cancer have risen more rapidly than ates of lung squamous cell cancer over the past 2 decades. METHODS: A control study was carried out to assess the impact of long-term filter ette smoking on the risk of squamous cell carcinoma (SCC) and carcinoma (AC) of the lung. RESULTS: Odds ratios for SCC among subjects who moked only filter cigarettes were reduced relative to lifetime nonfilter ette smokers by 30% for men and by 60% for women, but no risk reduction was ved for AC of the lung. CONCLUSION: The predominance of AC over SCC may be n part to the fact that smokers of very low yield cigarettes tend to usate for the lower nicotine levels by inhaling more deeply and frequently, ng to greater exposure of the peripheral lung to the carcinogens in tobacco, and in part to the increased concentration of nitrosamines that rentially produce AC in laboratory animals.

9245665, UI: 97394846

st J Med 1997 Mar; 166(3):189-94

an-American smokers and cancers of the lung and of the upper respiratory igestive tracts. Is menthol part of the puzzle?

rdson TL

tment of Internal Medicine, University of Colorado Health Sciences Center, USA.

revalence of cigarette smoking is higher among African Americans than among s. African Americans have higher rates of lung cancer than whites, although

smoke fewer cigarettes. To explore this black-white difference in lung r rates, I examine various aspects of tobacco use in African-American rs, including the age of initiation of smoking, quantity of cigarettes d, quit rates, level of nicotine dependence, biochemical differences, and preferences, specifically menthol brand cigarettes. I also review briefly equelae of patterns of tobacco use, including rates of lung and other co-related cancers. A preference for mentholated cigarettes by African cans is well documented and is one of the most striking differences between an-American and white smokers. Menthol brand preference has been tigated in an attempt to explain the black-white differences in rates of rs of the lungs and the upper respiratory and digestive tracts. Also, es have evaluated smoking behavior both with and without menthol and have citly examined the question of whether menthol use helps explain the -white difference in lung cancer rates. The results of these studies are so nconclusive with regard to the use of menthol and the risk of lung cancer oping. I provide practical suggestions for clinicians in counseling an-American smokers to quit smoking and to maintain a nonsmoking status.

cation Types: w w literature

9143194, UI: 97288211

ncer Causes Control 1997 Jan;8(1):13-24

ry and tobacco risk factors for adult onset glioma in the San Francisco Bay

, Wrensch M, Miike R

iment of Epidemiology and Biostatistics, University of California, San isco 94143-0560, USA.

oles of diet and tobacco in the etiology of primary brain cancer are oversial. In this report, we compare dietary and cigarette smoking ries among 434 adults newly diagnosed with glioma in the San Francisco Bay (California, USA) between 1991 and 1994 with frequency age, gender, and city-matched population-based controls. Data were obtained on use of in supplements and mean weekly consumption of each of 24 food groups. ted for age, family income, and education, for both men and women, cases igher mean weekly consumption of cured meats and other cured foods, lower mption of high vitamin A and C fruits and vegetables, and higher average

oth men and women, but there has been a much sharper incidence among females the mid-1970s. An examination of age-specific incidence rates by birth t and the results from age-period-cohort modeling indicate that incidences I three major histologic types of lung cancer in the recent birth cohorts r have started decreasing (squamous cell carcinoma) or shown a clear tion in the rate of increase (adenocarcinoma and small cell carcinoma). study, however, did not indicate an increase of bronchoalveolar lung noma, which was reported by other clinically based studies. CONCLUSION. the overall age-adjusted incidence rates showed different incidence erns for different histologic types of lung cancer, a decreasing or lized rate for all three major histologic types of lung cancer was observed cent birth cohorts in both males and females. The observed incidence rn is consistent with smoking trends over time including changes in smoking :lence and the consumption of low tar and filter cigarettes. It is expected if the current trend in tobacco smoking continues and if there are no major es in other risk factors for lung cancer, a forthcoming stabilization or ase in the rate of lung cancer incidence for all major histologic types uding adenocarcinoma) in both sexes in Connecticut could be anticipated.

8062189, UI: 94340504

lancer Epidemiol Biomarkers Prev 1992 Jan-Feb;1(2):103-7

c differences in the lung cancer risk associated with smoking.

rchand L, Wilkens LR, Kolonel LN

r Research Center of Hawaii, University of Hawaii, Honolulu 96813.

lity trends and ecological data strongly suggest that the lung cancer risk liated with smoking is greater among Hawaiians than among the other ethnic in Hawaii. The authors combined data from two consecutive ation-based case-control studies to formally test this hypothesis among 740 and 1616 controls. A multiple logistic regression analysis adjusting for years of smoking, occupation, education, and age revealed that Hawaiian, ino, and Caucasian male smokers were at 121%, 53%, and 46% greater risk for cancer than Japanese male smokers. These risk differences were stically significant, were consistent across sexes and histological types, ere not explained by the type of cigarettes, the level of inhalation, or by sterol or beta-carotene intake. Additionally, an increased lung cancer risk ated to smoking was observed among Chinese women. The possibility that dietary antioxidants and/or genetic risk factors are responsible for these c differences needs to be investigated.

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: 1306091, UI: 93306146
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Stat Bull Metrop Insur Co 1991 Apr-Jun: 72(2):2-5 oaches for reducing cancer mortality in minorities.

e is an urgent need to reduce cancer mortality among black Americans and r minorities. Although much progress has been made, cancer statistics are as positive for these groups and the medically-underserved populations. ing Americans to change their behavior patterns -- particularly with regard to rette smoking--is vitally important. In this regard, physicians and other th care professionals, as well as public and government officials, must k out. Early detection of risk factors is also critical. Government has an gation to make Americans aware of how to reduce adverse health indicators of making individuals responsible for their own life as well as for the s of their family members.

: 2063259, UI: 91289227

Am J Public Health 1988 Aug; 78(8): 986-7

hol cigarettes and esophageal cancer.

rt JR, Kabat GC

ication Types:

ivan LW

: 3389443, UI: 88267364

Cancer 1988 Jul 1;62(1):6-14

or tobacco smoke pollution. A major risk factor for both breast and lung or?

on AW

conmental hazards is an unusually early age at onset of disease. Animal ies have shown age effects of this type with co-carcinogens. A clearcut ile of a downward age shift in humans is provided by a study of the imption of alcoholic beverages and cigarettes in women with oral concer, if data on 145 white females with intraoral cancer, and 1973 non-neoplastic rols from patients seen at Roswell Park Memorial Institute between 1957 and it can be shown that exposure to both alcohol and tobacco can lead to to foral cancer 15 or more years earlier than would occur in women who do use either alcohol or tobacco. Exposure to smoking only produces a smaller shift, but exposure to alcohol only does not produce any clear shift in age nset. Implications for co-carcinogenesis and for early detection of arcinogens in the environment are suggested.

: 1012647, UI: 77101275

Jatl Cancer Inst Monogr 1968 Jun; 28:35-40

of filter digarettes on the risk of lung cancer.

a TD

: 5671411, UI: 68393359

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